cow's milk. An infant is susceptible to absorption of macromolecules (both microbes and food antigens) in the first year of life. Breast milk may provide this immature host with passive protection at this critical period, as well as with an excellent hypoallergenic source of nutrition.

STANLEY P. GALANT, MD

REFERENCES

Goldman AS, Smith CW: Host resistance factors in human milk. J Pediatr 82:1082-1090, Jun 1973 Gerrard JW: Breast feeding—second thoughts. Pediatrics 54: 757-764, Dec 1974

Pitt J: Breast milk leukocytes. Pediatrics 58:769-770, Nov 1976

Mucocutaneous Lymph Node Syndrome

MUCOCUTANEOUS LYMPH NODE SYNDROME (MLNS) is a recently recognized disease of unknown etiology which has a mortality of 1 to 2 percent. It has occurred with increasing frequency in Japan and is now being recognized in many countries throughout the world, including the United States. It is characterized by fever which persists for more than five days; an erythematous skin eruption; conjunctival congestion; dry fissured red lips; red tongue, palms, and soles; nonpurulent lymphadenopathy; diarrhea; arthralgia and aseptic meningitis. Additional striking features which occur less frequently include carditis; pericarditis; aneurysmal dilatation and thrombosis of coronary arteries; and sudden death. Fatal cases have features which are indistinguishable from infantile polyarteritis nodosa. In some surviving infants there is the transient appearance of coronary aneurysms which subsequently disappear. A few patients have recurrent disease but the vast majority appear to recover completely. Many cases mimic the Stevens-Johnson syndrome. MLNs may actually represent a subdivision of the Stevens-Johnson syndrome as well as one subdivision of infantile polyarteritis nodosa. It has many features of an infectious process and the possibility that the etiologic agent may be viral or rickettsial is currently under investigation in a number of laboratories.

During a collaborative investigation of Japanese children with the syndrome in association with Dr. Kusakawa it was found that early in the course of MLNs there is a 3- to 4-fold elevation of serum IgE level which peaks at 7 to 12 days after onset of the disease and declines over the

ensuing 30 to 60 days. This suggests an element of hypersensitivity in the disease, and the vasculitis which commonly occurs suggests a similarity to serum sickness. There is also a significant elevation of total serum IgM but the peak is smaller and later than that of IgE. It is the first nonparasitic acute febrile disease which has been shown to be regularly accompanied by an elevation of serum IgE level and it is the first disease in which a marked IgE response has been shown to precede a significant IgM response. It appears that in patients who have recurrence of the disease the IgE level becomes elevated again, and in patients who have persistent active coronary arteritis IgE levels remain high. Serial IgE levels, therefore, may prove of diagnostic value and help in recognizing continued disease activity.

DOUGLAS C. HEINER, MD

REFERENCES

Kawasaki T, Kosaki F, Okawa S, et al: A new infantile acute febrile mucocutaneous lymph node syndrome (MLNS) prevailing in Japan. Pediatrics 54:271-276, Sep 1974

Melish ME, Hicks RM, Larson EJ: Mucocutaneous lymph node syndrome in the United States. Am J Dis Child 130:599-607,

Kusakawa, S, Heiner DC: Elevated levels of immunoglobulin E in the acute febrile mucocutaneous lymph node syndrome. Pediatr Res 10:108-111, Feb 1976

The Hazard of IgE Mediated Allergic **Reaction After Blood Transfusion**

RECENT REPORTS have confirmed the potential risk to infants of passively transferred IgE following exchange transfusion. A single unit of blood or plasma containing a high titer of IgE or an exchange transfusion with blood containing normal amounts of IgE results in a 70 percent uptake by basophils and mast cells throughout the body within 24 hours. By 48 hours maximal fixation has occurred. The skin fixed IgE is shown to persist in significant quantities for at least 31 days. Consequently, the IgE infused in conjunction with a transfusion can place an infant at risk of an anaphylactic reaction for at least one month.

> ROBERT N. HAMBURGER, MD MICHAEL H. MELLON, MD MACIEJ TOMASZEWSKI, MD H. ALICE ORGEL, MD

REFERENCES

Mellon MH, Orgel HA, Hamburger RN: The fate of IgE in infants following exchange transfusion. Clin Res 25:182A, Feb 1977

Mellon MH, Tomaszewski MF, Orgel HA, et al: Serum levels and skin fixation of transfused IgE in infants. Ped Res 11:490, Apr 1977

Ramirez MA: Horse asthma following blood transfusion—Report of case. JAMA 73:984-985, Sep 27, 1919